

**STATUS OF CLAIMS**

**Claims 1-33 (previously canceled)**

**Claim 34 (previously amended)** The method of claim 69 wherein the compound is determined to be an inverse agonist to said receptor.

**Claims 35-39 (previously canceled)**

**Claim 40 (previously amended)** The method of claim 70 wherein the compound is determined to be an inverse agonist to said receptor.

**Claim 45 (previously amended)** The method of claim 69 wherein the third intracellular loop of the receptor of step (a) comprises the following sequence:

X1BBHyX2

Wherein X1 is an amino acid; B is a basic amino acid; Hy is a hydrophobic amino acid; and X2 is an amino acid.

**Claim 46 (original)** The method of claim 45 wherein X1 is glycine.

**Claim 47 (original)** The method of claim 45 wherein X1 is lysine.

**Claim 48 (original)** The method of claim 45 wherein Hy is alanine.

**Claim 49 (original)** The method of claim 45 wherein X2 is lysine.

**Claim 50 (original)** The method of claim 45 wherein X2 is arginine.

**Claim 51 (original)** The method of claim 45 wherein X2 is glutamic acid.

**Claim 52 (previously amended)** The method of claim 69 wherein the second intracellular loop of the receptor of step (b) comprises the following sequence:

XRY

wherein X can be any amino acid other than aspartic acid; R is arginine; and Y is tyrosine.

**Claim 53 (previously amended)** The method of claim 70 wherein the third intracellular loop of the receptor of step (a) comprises the following sequence:

X1BBHyX2

wherein X1 is an amino acid; B is a basic amino acid; Hy is a hydrophobic amino acid; and X2 is an amino acid.

Claim 54 (**original**) The method of claim 53 wherein X1 is glycine.

Claims 55 (**original**) The method of claim 53 wherein X1 is lysine.

Claim 56 (**original**) The method of claim 53 wherein Hy is alanine.

Claim 57 (**original**) The method of claim 53 wherein X2 is lysine.

Claim 58 (**original**) The method of claim 53 wherein X2 is arginine.

Claim 59 (**original**) The method of claim 53 wherein X2 is glutamic acid.

Claim 60 (**previously amended**) The method of claim 70 wherein the second intracellular loop of the receptor of step (a) comprises the following sequence:

XRY

wherein X can be any amino acid other than aspartic acid; R is arginine; and Y is tyrosine.

Claim 61 (**original**) The method of claim 45 wherein the sequence X1BBHyX2 is an endogenous sequence.

Claim 62 (**original**) The method of claim 52 wherein the sequence XRY is an endogenous sequence.

Claim 63 (**previously amended**) The method of claim 69 wherein said mammal of step (d) is a human.

Claim 64 (**previously amended**) The method of claim 70 wherein said mammal of step (d) is a human.

Claim 65 (**previously amended**) The method of claim 69 wherein said mammal of step (d) is a non-human.

Claim 66 (**previously amended**) The method of claim 70 wherein said mammal of step (d) is a non-human.

Claim 67 (**previously amended**) The method of claim 69 wherein said physiological function is an abnormal physiological function.

Claim 68 (**previously amended**) The method of claim 70 wherein said physiological function is an abnormal physiological function.

Claim 69 (**previously added**) A method for directly identifying a non-endogenous candidate compound as an agonist or an inverse agonist to an endogenous G protein coupled

receptor (GPCR), wherein a location of expression of said receptor in a mammalian tissue source is known and said receptor has been correlated with at least one mammalian physiological function and wherein an endogenous ligand for said receptor has not been identified, said method comprising the steps of:

- (a) subjecting said GPCR to constitutive receptor activation to create a constitutively activated GPCR;
- (b) contacting the non-endogenous candidate compound with said constitutively activated GPCR;
- (c) identifying said non-endogenous candidate compound as an inverse agonist or an agonist to said constitutively activated GPCR ~~by measuring at least a 30% difference in a reporter signal induced by said contacted compound as compared with a reporter signal in the absence of said contacted compound.~~

Claim 70 (**currently amended**) A method for directly identifying a non-endogenous candidate compound as an agonist or an inverse agonist to an endogenous constitutively activated active G protein coupled receptor (GPCR), wherein a location of expression of said receptor in a mammalian tissue source is known and said receptor has been correlated with at least one mammalian physiological function and wherein an endogenous ligand for said receptor has not been identified, said method comprising the steps of:

- (a) contacting the non-endogenous candidate compound with said constitutively activated active GPCR;
- (b) identifying said non-endogenous candidate compound as an inverse agonist or an agonist to said constitutively activated active GPCR ~~by measuring at least a 30% difference in a reporter signal induced by said contacted compound as compared with a reporter signal in the absence of said contacted compound.~~

Claim 71 (**previously added**) A compound directly identified by the method of claim 69.

Claim 72 (**previously added**) A compound directly identified by the method of claim 70.

Claim 73 (**previously added**) A pharmaceutical composition comprising the compound of claim 71.